

Cancer Risk from Incidental Ingestion Exposures to PAHs Associated with Coal-Tar-Sealed Pavement

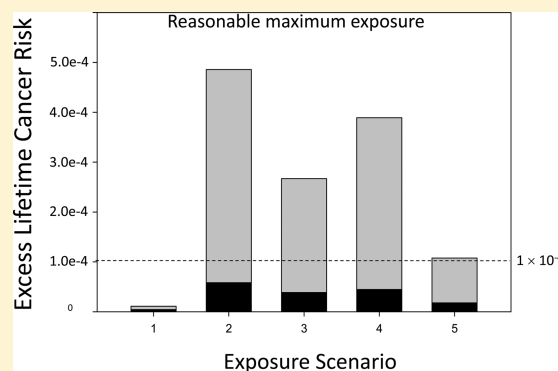
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S Supporting Information

ABSTRACT: Recent (2009–10) studies documented significantly higher concentrations of polycyclic aromatic hydrocarbons (PAHs) in settled house dust in living spaces and soil adjacent to parking lots sealed with coal-tar-based products. To date, no studies have examined the potential human health effects of PAHs from these products in dust and soil. Here we present the results of an analysis of potential cancer risk associated with incidental ingestion exposures to PAHs in settings near coal-tar-sealed pavement. Exposures to benzo[*a*]pyrene equivalents were characterized across five scenarios. The central tendency estimate of excess cancer risk resulting from lifetime exposures to soil and dust from nondietary ingestion in these settings exceeded 1×10^{-4} , as determined using deterministic and probabilistic methods. Soil was the primary driver of risk, but according to probabilistic calculations, reasonable maximum exposure to affected house dust in the first 6 years of life was sufficient to generate an estimated excess lifetime cancer risk of 6×10^{-5} . Our results indicate that the presence of coal-tar-based pavement sealants is associated with significant increases in estimated excess lifetime cancer risk for nearby residents. Much of this calculated excess risk arises from exposures to PAHs in early childhood (i.e., 0–6 years of age).



INTRODUCTION

The presence of coal-tar-based sealants on asphalt parking lots is associated with elevated concentrations of polycyclic aromatic hydrocarbons (PAHs) in the surrounding environment.^{1–6} Sealcoat is a black, shiny substance sprayed or painted on the asphalt pavement of parking lots, driveways, and playgrounds to improve appearance and protect the underlying asphalt. An estimated 85 million gallons (320 million liters) of coal-tar-based sealant are applied to pavement each year,⁷ primarily east of the Continental Divide in the U.S. and parts of Canada.^{4,8} Coal-tar-based pavement sealants are 15–35% coal-tar pitch, which has been classified as a human carcinogen (IARC Group 1).⁹ PAHs are the major constituents of coal-tar pitch,¹⁰ and commercially available coal-tar-based sealants contain on the order of 50 000–100 000 mg/kg PAHs [sum of the 16 U.S. Environmental Protection Agency (USEPA) Priority Pollutant PAHs (ΣPAH_{16})].^{7,11} Over time, the dried sealant is abraded from pavement surfaces, and the resulting mobile particles can be transported into nearby environmental compartments.^{7,12}

Coal-tar-based pavement sealants are the predominant source of PAHs in the sediment of many urban and suburban lakes, especially areas where population is rapidly growing.^{3,13} Coal-tar-based sealants are associated with deleterious effects on local ecosystems, including decreases in species richness and abundance among benthic invertebrates,^{14,15} slower growth and

impaired swimming behaviors in salamanders,¹⁶ and impaired growth and development of frogs.¹⁷ PAHs from coal-tar-based pavement sealants also contaminate environmental media that are relevant to human exposures. In a study of 23 apartments in Austin, Texas, the median concentration of ΣPAH_{16} in settled house dust (SHD) in residences adjacent to coal-tar-sealed asphalt (CSA) parking lots was 31 times higher than in SHD in apartments adjacent to unsealed asphalt (UA) lots.¹⁸ The presence or absence of coal-tar-based sealants on the adjacent lot explained 48% of the variance in PAH concentrations measured in SHD.¹⁸ Elevated PAH concentrations also have been reported for soil adjacent to CSA lots relative to soil adjacent to UA lots.^{2,4} Hereinafter, soil and SHD near CSA or UA parking lots are described as “CSA-affected” or “UA-affected”, respectively.

Exposure to PAHs is linked to increased risk for multiple cancer types, including lung, skin, bladder, respiratory, and urinary tract.¹⁹ These studies have mostly examined inhalation exposure at smelting plants, foundries, and similar industrial settings. The carcinogenic properties of tobacco smoke are attributed, in part, to the presence of PAHs.²⁰ Aside from

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smoking, nonoccupational exposures to PAHs are believed to occur primarily through dietary ingestion.²¹ In the interest of understanding aggregate doses, several studies have characterized the presence of PAHs in a wide array of foodstuffs in different countries, including the U.S., as reviewed in Ramesh et al. (2004).²¹ Seven PAHs—benz[*a*]anthracene, benzo[*k*]fluoranthene, benzo[*b*]fluoranthene, benzo[*a*]pyrene (BaP), chrysene, dibenz[*a,h*]anthracene (diBaH_A), and indeno[123-*cd*]pyrene—have been classified by the USEPA as probable human carcinogens (B2 PAHs).

Nondietary ingestion (incidental ingestion of soil and SHD) is a pathway for exposure to numerous chemicals, including lead, pesticides, polychlorinated dioxins and furans, polycyclic aromatic hydrocarbons (PAHs), especially in children.^{22,23} Many sources and activities are hypothesized to contribute PAHs to SHD, including cooking, smoking, vehicle exhaust, and indoor heating.^{24,25} These exposures have been characterized as minor relative to those associated with dietary ingestion;^{26,27} however, recent research indicates that in CSA-affected residences, nondietary ingestion of PAHs likely exceeds dietary ingestion.²⁸

To date (November 2012), the authors are not aware of any published studies that have assessed the potential risks to human health associated with the elevated concentrations of PAHs measured in CSA-affected environments. The objective of the current study was to examine and compare exposure to and risk arising from ingestion of B2 PAHs in SHD and soil in settings adjacent to CSA and UA parking lots. Standard deterministic risk-assessment techniques were used to estimate B2 PAH doses and associated excess lifetime cancer risk (ELCR) for five exposure scenarios spanning childhood, adolescence, and adulthood, and probabilistic risk calculations were conducted for three of these scenarios.²⁹

METHODS

This risk assessment focuses on the B2 PAHs. Each of these compounds has been assigned a potency factor (RPF) relative to the potency of BaP, ranging from 0.001 for chrysene to 1 for diBaH_A and BaP.³⁰ Ingestion dose estimates are presented for BaP equivalents (BaPEQ), computed as the sum of the product of the concentration of each B2 PAH and its RPF. Bioavailability is assumed to be 100%.

As noted in ref 18, analytical difficulties with diBaH_A resulted in nondetections in all but one SHD sample collected for that study. Thus, diBaH_A is not included here in any computations of BaPEQ in SHD or soil. Estimates of dose including diBaH_A at the limit of detection divided by two (not shown) indicate that it likely accounted for no more than 5–7% of the total dose of BaPEQ. By comparison, BaP accounted for 72–73% of BaPEQ in SHD samples, and 76–77% in soil samples.

Concentrations of BaPEQ in Dust and Soil. Data on PAHs in SHD used for this analysis were published previously.¹⁸ In that study, SHD and parking lot dust were sampled for 23 ground-floor apartments in Austin, Texas. The parking lot surface adjacent to the apartment complexes was CSA ($n = 11$), UA ($n = 7$), asphalt-based sealant over asphalt pavement ($n = 3$), or unsealed concrete ($n = 2$). For this analysis, doses and risk associated with residences adjacent to UA parking lots were considered relative to those adjacent to CSA parking lots. BaP concentrations in CSA-affected SHD were high (median and maximum of 4.5 and 24.2 $\mu\text{g/g}$, respectively) relative to those reported in most parts of the U.S. where coal-tar-based sealcoat is not used (e.g., California:

median and maximum of 0.04 and 1.0 $\mu\text{g/g}$, respectively; Arizona: median and maximum of 0.06 and 0.07 $\mu\text{g/g}$, respectively²⁵). We computed BaPEQ for data presented in,¹⁸ concentrations of BaPEQ in SHD in apartments adjacent to CSA parking lots (8.1 $\mu\text{g/g}$, geometric mean) were significantly higher than those in apartments adjacent to UA lots (0.61 $\mu\text{g/g}$, geometric mean) ($p = 0.002$, Mann–Whitney–Wilcoxon). Risk-assessment guidance recommends the use of the 95% upper confidence limit of the arithmetic mean,²⁹ but high standard deviations in the data sets, normality testing in log-transformed data, and an emphasis on conservatism in dose and risk estimates dictated the decision to use geometric means of these data to represent the BaPEQ exposure concentration in deterministic calculations.

Dust loading was computed for each location sampled in ref. 18 (Supporting Information Table S1). Loading of BaPEQ in the dust is significantly higher in residences adjacent to CSA pavement (medians of 15.7 $\mu\text{g}/\text{m}^2$ CSA vs 0.63 $\mu\text{g}/\text{m}^2$ UA; $p = 0.01$, Mann–Whitney–Wilcoxon). Total dust loading is higher in the CSA group relative to the UA group (medians of 346 and 72.3 $\mu\text{g}/\text{cm}^2$, respectively), but the difference was not significant ($p = 0.365$, Mann–Whitney–Wilcoxon). However, one data point in the UA SHD data set is an outlier (884 $\mu\text{g}/\text{cm}^2$) more than 4 times larger than all other data points and after removal of this data point, CSA settings have significantly higher dust loadings than UA settings ($p = 0.043$, Student's *t* test; data passed normality testing after elimination of the outlier). One issue that could not be resolved in this analysis is the relative importance of flooring type, because some samples were collected in combinations of bare and carpeted flooring.

Data for PAHs in CSA- and UA-affected soils are available for samples from New Hampshire (UA $n = 1$, CSA $n = 5$)² and suburban Chicago (UA $n = 2$, CSA $n = 2$).⁴ Concentrations of BaP in UA-affected soils ranged from below detection limit to 0.7 $\mu\text{g/g}$. These are consistent with background concentrations reported for U.S. soils of up to 1.3 $\mu\text{g/g}$,¹⁹ and somewhat higher than those reported for soil samples collected in remote areas around the world (range <0.0001 to 0.386 $\mu\text{g/g}$).³¹ Concentrations of BaP in CSA-affected soils were substantially higher, ranging from 2.98 to 29.2 $\mu\text{g/g}$.^{2,4} Concentrations of BaP in dust on pavement with coal-tar-based sealant are typically in the 100s of $\mu\text{g/g}$.^{2,18} Concentrations of BaP in the 100s of $\mu\text{g/g}$ in soil are typical of those in soils at manufactured gas sites and wood preservative sites,^{32,33} some of which have been classified as Superfund sites (<http://www.epa.gov/region5/cleanup/mgp.htm>). Geometric mean BaPEQ soil concentrations for CSA-affected settings were 12.4 μg BaPEQ/g soil, and for UA-affected settings were 0.19 μg BaPEQ/g soil.

Deterministic and Probabilistic Estimates of Dose and Excess Lifetime Cancer Risk. Doses of BaPEQ were estimated using the standard equation (eq 1) included in the Risk Assessment Guidance for Superfund, Part A.²⁹ Exposure assumptions for both deterministic and probabilistic risk calculations are given in Supporting Information Table S2.

$$\text{dose} = \frac{\text{Cm} \times \text{CF} \times \text{IR} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}} \quad (1)$$

where Cm is the concentration of BaPEQ in the dust, soil, or both, CF is the conversion factor, IR is ingestion rate, EF is exposure frequency, ED is exposure duration, BW is body weight, and AT is averaging time.

Table 1. Excess Lifetime Cancer Risk (ELCR) Estimates for Central Tendency (CTE) and Reasonable Maximum (RME) Exposures in Five Scenarios for Carcinogenic Polycyclic Aromatic Hydrocarbons by Ingestion of Settled House Dust, Soil, and Both Media^a

scenario	age of exposure (years of age)		settled house dust only		soil only		dust and soil	
	UA	CSA	CTE	RME	CTE	RME	CTE	RME
1	0–70	N/A	1.5×10^{-6}	4.4×10^{-6}	1.4×10^{-6}	6.7×10^{-6}	2.9×10^{-6}	1.1×10^{-5}
2	N/A	0–70	2.0×10^{-5}	5.8×10^{-5}	8.9×10^{-5}	4.3×10^{-4}	1.1×10^{-4}	4.9×10^{-4}
3	6–70	0–<6	1.1×10^{-5}	3.8×10^{-5}	2.9×10^{-5}	2.3×10^{-4}	4.0×10^{-5}	2.7×10^{-4}
4	18–70	0–<18	1.4×10^{-5}	4.4×10^{-5}	4.7×10^{-5}	3.4×10^{-4}	6.1×10^{-5}	3.9×10^{-4}
5	0–<18	18–70	8.2×10^{-6}	1.8×10^{-5}	4.3×10^{-5}	9.0×10^{-5}	5.1×10^{-5}	1.1×10^{-4}

^aUA, unsealed asphalt pavement; CSA, coal-tar-sealed asphalt pavement; N/A, not applicable.

The geometric mean BaPEQ for SHD and soil were used as point estimates for deterministic dose and risk calculations. Lognormal distributions based on data from refs 2,4,18 were developed for probabilistic calculations [UA soil: mean 0.423 $\mu\text{g/g}$ (standard deviation (sd) = 0.523), CSA soil: mean 15.8 $\mu\text{g/g}$ (sd = 11.9); UA SHD: mean 1.10 $\mu\text{g/g}$ (sd = 1.08), CSA SHD: mean 11.4 $\mu\text{g/g}$ (sd = 9.41)]. Lognormal distributions and corresponding geometric means were chosen to reflect the frequent observation of distributions of this type in environmental contaminant concentrations.

For deterministic calculations of SHD ingestion, we used recently published SHD intake rates for children determined using the Stochastic Human Exposure and Dose Simulation (SHEDS) model for multimedia pollutants.³⁴ The SHEDS model addresses two pathways of exposure to dust: direct ingestion of SHD from hand-to-mouth contact, and indirect ingestion resulting from mouth contact with inanimate objects such as toys (especially relevant for preschool children). The model takes into account the importance of SHD loading, a strong predictor of blood lead levels related to dust-mediated exposure. The model relies on the Consolidated Human Activity Database, which has activity diaries for over 22 000 individuals.³⁵ We employed the mean SHD IR estimate from ref. 34 of 27 mg/day (rounded to two significant figures to account for the inherent uncertainty of the model) for children 3–<6 years of age as a central tendency estimate (CTE) of exposure for children 0–6 years of age, and the 95th percentile values from³⁴ as a reasonable maximum estimate (RME) of exposure. For individuals older than 6 years of age, who are expected to be away from the home for much of the day, we used one-half of the early childhood CTE dust IR (13 mg/day), and 27 mg/day as the RME dust IR. Few data are available for SHD IRs for adults, but previous risk assessments have employed adult SHD IRs of 20 and 50 mg/day,^{22,36} higher than the IRs used in this analysis. The distribution of child IRs for SHD was adapted from ref. 34 (mean = 27 mg/day, sd = 40, log-normal) for probabilistic dose and risk calculations, and a similarly shaped distribution was postulated for SHD IR for 6–70 years of age (mean = 13.3 mg/day, sd = 19.6, log-normal).³⁴

For deterministic calculations of soil ingestion, default IRs from the Exposure Factors Handbooks and the Child Specific Exposure Factors Handbook,^{37,38} with some minor modifications, were used. For persons of all ages, 50 mg/day was used for the CTE soil IR, and the RME IRs used were 400 mg/day from 1–13 years of age and 100 mg/day from 13–70 years of age.

For a distribution for soil IRs for children 0–<13 years of age, we used data generated by the SHEDS model that indicated an arithmetic mean of 60.6 mg/day, sd of 80.5 mg/day.³⁹ These values are similar to those from a recent review of

all published tracer studies on soil ingestion by children, in which the arithmetic mean was estimated at 63 mg/day, with a median of 27 mg/day and a 95th percentile of 195 mg/day.³⁹ The SHEDS model result was used as the basis for probabilistic calculations of dose and risk in children. For children and adults 13–70 years of age, the arithmetic mean of all available soil ingestion rates from tracer studies was 46 mg/day (rounded to 50 mg/day in deterministic calculations).³⁹ A distribution similar to that for soil ingestion in children was postulated, and an appropriate standard deviation was calculated for use in a Monte Carlo analysis (<http://www.epa.gov/oswer/riskassessment/rags3adt/index.htm>). Adult IRs have been updated in the most recent (2011) version of the Exposure Factors Handbook to indicate a central tendency for adults of 20 mg/day for the soil IR and 30 mg/day for the dust IR.⁴⁰ These values rely on relative proportions of soil and dust ingestion for children, and thus we have chosen to retain the value of 50 mg/day (i.e., 46 mg/day, rounded to one significant digit) from the previous Handbook, which also is the value indicated in the current Handbook for adults 18–21 years of age.⁴⁰ Recalculation of risk estimates using soil and dust ingestion rates in the 2011 version of the Handbook do not change the overall conclusions of this assessment.

Body weight distributions were obtained from a recent (2007) analysis of the National Health and Nutrition Examination Survey (NHANES) data set.⁴¹ Exposure frequency was set at 365 days/year in both deterministic and probabilistic calculations.

Exposure Scenarios. Five scenarios that describe exposures to combinations of UA- and CSA-affected SHD and soil were used (Table 1): exposures in UA-adjacent spaces (UA exposures) during a 70-year lifetime (scenario 1); exposure in CSA-adjacent spaces (CSA exposures) during a 70-year lifetime (scenario 2); CSA exposures during 0–<6 years of age followed by UA exposures during 6–70 years of age (scenario 3); CSA exposures during childhood (0–<18 years of age) followed by UA exposures during adulthood (18–70 years of age, scenario 4); and UA exposures during 0–<18 years of age followed by CSA exposures during adulthood (18–70 years of age, scenario 5). Incremental ELCR values for timeframes of 1 year from 0 to 18 years of age and of 1 year from 18 to 70 years of age were summed to arrive at a lifetime ELCR value for each scenario. Exposure to UA-affected environments during a 70-year lifetime (Scenario 1) was assumed to represent urban background for the purpose of evaluating the potential differences in risks associated with exposure to CSA-affected media. Scenario 1 considers lifetime exposures to SHD and soil not affected by PAHs associated with CSA pavement, and thus represents a reasonable measure of urban background.

For the probabilistic calculations, Monte Carlo simulations were performed for 10 000 trials. These simulations were conducted only for scenarios covering lifetime exposures to UA environments (scenario 1), lifetime exposures to CSA environments (scenario 2), and exposures to CSA-affected media in the first 6 years of life (scenario 3).

Estimation of Excess Lifetime Cancer Risk. The ELCR from exposure to a chemical is described in terms of the probability that an exposed individual will develop cancer by age 70 because of that exposure.⁴² Estimates of BaPEQ dose were multiplied by the oral cancer slope factor for BaP of 7.3 per mg/kg/day.⁴³ For single-year calculations of risk (0–18 years of age), the slope factor was divided by 70, and for calculation of risk for adulthood (18–70 years of age), it was divided by (70/52); risk estimates were generated by summing yearly risks from 0–18 years of age and during adulthood (i.e., 18–70 years of age). In general, the USEPA considers excess cancer risks less than 1×10^{-6} so small as to be negligible (i.e., de minimus), and those greater than 1×10^{-4} to be sufficiently large that some sort of remediation is desirable.⁴² Excess cancer risks between 1×10^{-6} and 1×10^{-4} generally are considered to be acceptable, although this is evaluated on a case-by-case basis and the USEPA may determine that risks lower than 1×10^{-4} are not sufficiently protective and warrant remedial action.⁴²

RESULTS

Deterministic Dose Estimates. Estimated lifetime CTE BaPEQ dose from ingestion of SHD and soil in CSA-affected settings was 38 times greater than that estimated for UA-affected settings (Supporting Information Table S3). Maximum doses occur at young ages (Figure 1), when body weights are lower and ingestion rates are higher than later in life (Supporting Information Table S3). About 50% of the total estimated RME lifetime dose occurs during 0–<6 years of age, and about 80% occurs during 0–<18 years of age. Doses of BaPEQ for ingestion of CSA-affected soil were greater than those for CSA-affected SHD (Figure 1), comprising about 80% of the aggregate (soil + SHD) lifetime dose. The difference arises because BaPEQ concentrations and IRs are higher for CSA-affected soil than for CSA-affected SHD (Supporting Information Table S2). The CTE lifetime dose from CSA-affected SHD alone, however, is not insubstantial, exceeding the lifetime aggregate dose in UA-affected settings by a factor of 7. The RME lifetime aggregate dose estimate for CSA-affected settings is about 4.5 times higher than the CTE lifetime aggregate dose estimate.

Risk Estimates. Deterministic estimates of ELCR were calculated for the five exposure scenarios (Table 1, Figure 2). Under scenario 1 conditions (urban background), soil is estimated to contribute about one-half (48%) of the aggregate (SHD + soil) CTE estimate of ELCR of 2.9×10^{-6} and the majority (61%) of the RME estimate of 1.1×10^{-5} .

Estimated aggregate CTE ELCR for lifetime exposure to CSA-affected settings (1.1×10^{-4} ; scenario 2) was 38 times higher than urban background (scenario 1) (Figure 2). About 36% of the increased ELCR attributable to ingestion of CSA-affected SHD and soil occurs during exposures during the first 6 years of life (scenario 3), when IRs are highest and body weights are lowest, and 56% occurs during the first 18 years of life (scenario 4). The RME ELCRs were from 2.2 to 6.8 times higher than CTE ELCRs across all CSA-affected scenarios (2–

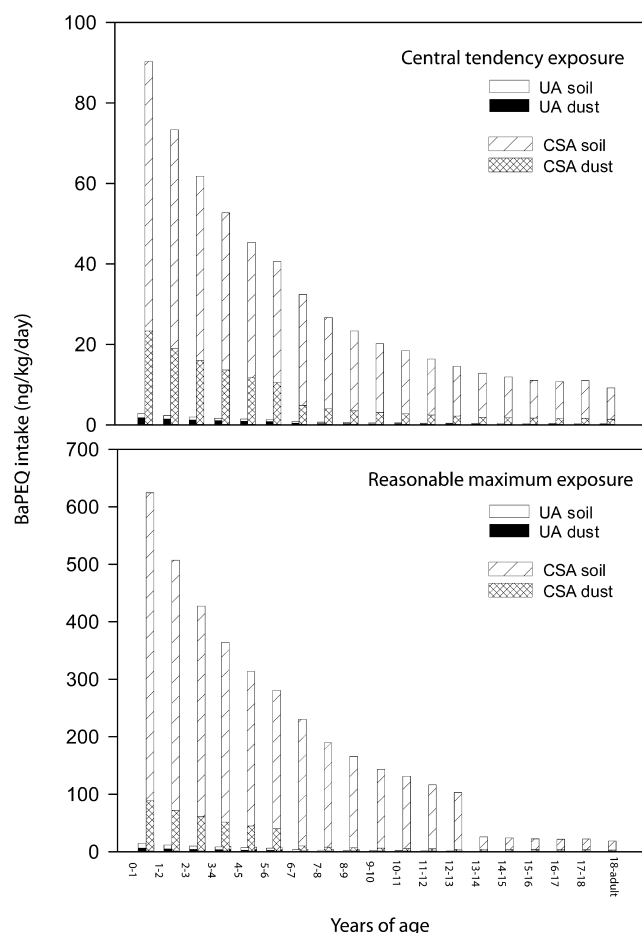


Figure 1. Aggregate doses of benzo[*a*]pyrene equivalents (BaPEQ) (ng/kg/day) from settled house dust and soil in settings adjacent to unsealed asphalt and coal tar-sealed asphalt pavement (UA and CSA, respectively) by year for central tendency and reasonable maximum exposures. Adult years (i.e., 18–70 years of age) are noted as “18-adult.”

5), and the difference was greatest for exposure to CSA-affected environments from 0–6 years of age (scenario 3) (Figure 2).

In this analysis, ingestion of CSA-affected soil is a more important driver of risk than ingestion of CSA-affected SHD. Ingestion of soil made up about one-half (48%) of ELCR in urban background settings, but made up 72 to 84% of ELCR in CSA-affected settings (Figure 2). Over a lifetime of exposure (scenario 2, CTE), ELCR is estimated to be about 64 times greater for persons who ingest CSA-affected soil relative to their counterparts who are exposed to background concentrations; the comparable difference for CSA-affected and unaffected SHD is a factor of 13. The CTE ELCR for soil alone approaches 1×10^{-4} , and the RME ELCR was estimated at 4.3×10^{-4} (Table 1). Much of the lifetime risk occurs during early childhood (0–<6 years of age, scenario 3) and all childhood (0–<18 years of age, scenario 4) exposures (33 and 53%, respectively). All RME scenarios in CSA-affected environments involving childhood exposure (scenarios 2–4) had ELCR values associated with ingestion of soil exceeding 1×10^{-4} .

Although SHD-mediated exposure to BaPEQ in CSA settings results in less risk compared to soil-mediated exposure, it nonetheless represents a substantial increase in risk over urban background exposure. This is a particularly important pathway of exposure for children. Even more of the lifetime risk

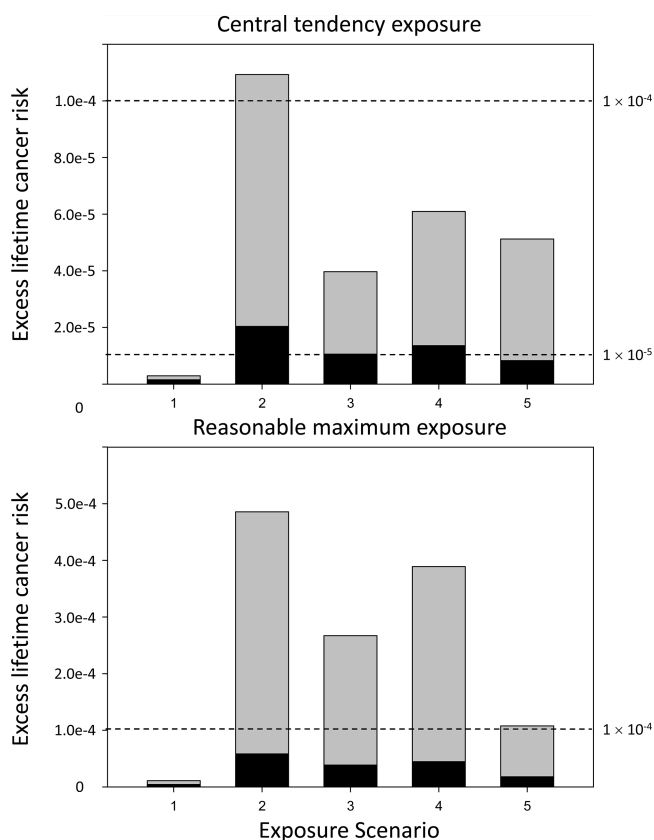


Figure 2. Deterministic excess lifetime cancer risk estimates for the five exposure scenarios described in Table 1 under central tendency and reasonable maximum exposure conditions. Risk attributable to dust is shown in black, and risk attributable to soil is shown in gray.

occurs during early childhood than it does for soil-mediated exposure, with 48 and 64% of the SHD-mediated risk occurring during the first 6 and 18 years of life, respectively. This difference results because the CTE IR for SHD is decreased to one-half its value at age 6 but the CTE IR for soil remains constant from 0–70 years of age (Supporting Information Table S2). All RME scenarios in CSA-affected environments (scenarios 2–5) had ELCR values for ingestion of SHD alone exceeding 1×10^{-5} but none exceeding 1×10^{-4} .

A probabilistic analysis (Monte Carlo) for scenarios 1, 2, and 3 yielded ELCR estimates in a range similar to those estimated deterministically (Table 2, Figure 3), where the 50th percentile statistic is treated as analogous to the CTE and the 95th percentile statistic is treated as analogous to the RME. As with deterministic estimates, probabilistic estimates for ELCR in CSA-affected settings for soil exposures (scenarios 2 and 3) were markedly higher than those for urban background settings (scenario 1) (Table 2). Probabilistic CTE ELCR estimates were

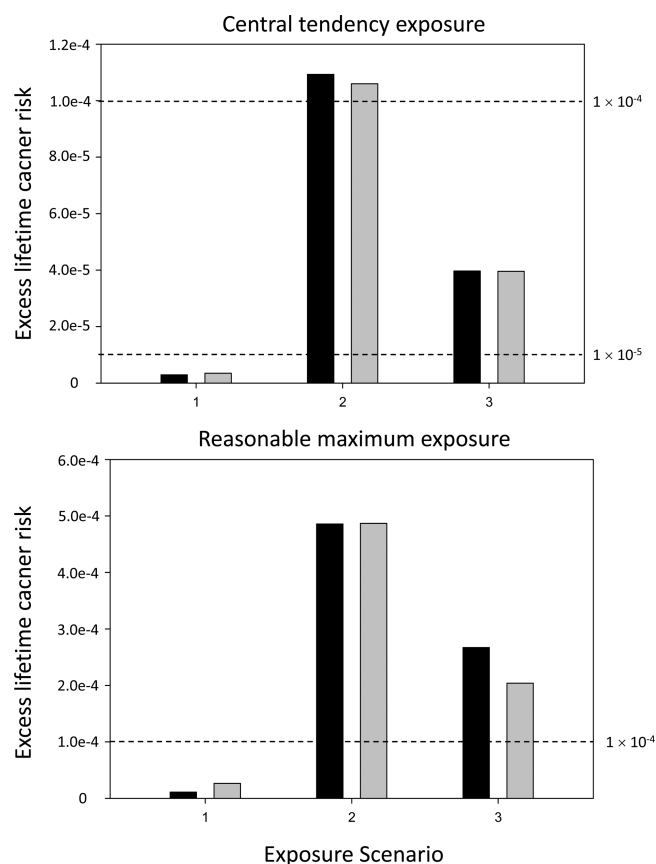


Figure 3. Comparison of deterministic and probabilistic estimates of excess lifetime cancer risk for three exposure scenarios for central tendency exposures (CTE) and reasonable maximum exposures (RME). Deterministic CTE estimates are analogous to 50th percentile probabilistic values, and deterministic RME estimates are analogous to 95th percentile probabilistic values. Black and gray bars depict deterministic and probabilistic risk estimates, respectively.

very similar to deterministic estimates (Table 1), within 21% for urban background (scenario 1) and identical for 70-year lifespan and the first 6 years of life (scenarios 2 and 3). Probabilistic 95th percentile ELCR estimates differed more from the deterministic estimates, exceeding the deterministic RME for urban background (scenario 1) by a factor of more than 2 and being less than it for the first 6 years of life (scenario 3) by 26%, but the probabilistic and deterministic RME estimates for a 70-year lifespan (scenario 2) were identical.

Sensitivity analyses for the probabilistic ELCR estimates indicate that the proportion of the variability in ELCR contributed by contaminant concentration and IR was different for each scenario (Table 3). For environments where ingestion of UA-affected media only was considered (scenario 1), BaPEQ concentration contributed most of the variability and IR

Table 2. Summary of Probabilistic Estimates (Monte Carlo Simulations, 10 000 runs, 50th Percentile Represents the Central Tendency Exposure and 95th Percentile Represents the Reasonable Maximum Exposure) of Excess Lifetime Cancer Risk for Exposure Scenarios 1–3

scenario	settled house dust only		soil only		dust and soil	
	50th	95th	50th	95th	50th	95th
1	1.2×10^{-6}	1.4×10^{-5}	1.1×10^{-6}	1.6×10^{-5}	3.5×10^{-6}	2.6×10^{-5}
2	1.8×10^{-5}	1.2×10^{-4}	7.3×10^{-5}	4.3×10^{-4}	1.1×10^{-4}	4.9×10^{-4}
3	8.3×10^{-6}	6.1×10^{-5}	2.4×10^{-5}	1.7×10^{-4}	4.0×10^{-5}	2.0×10^{-4}

Table 3. Proportion of the Variability in Estimates of Excess Lifetime Cancer Risk Contributed by Parameters Considered^{ab}

	scenario 1			scenario 2			scenario 3		
	dust alone	soil alone	dust and soil	dust alone	soil alone ^c	dust and soil ^c	dust alone	soil alone	dust and soil
[BaPEQ] _{UA dust}	0.71	--	0.33	--	--	--	0.03	--	--
[BaPEQ] _{CSA dust}	--	--	--	0.55	--	0.07	0.35	--	0.07
[BaPEQ] _{UA soil}	--	0.80	0.42	--	--	--	--	0.01	0.01
[BaPEQ] _{CSA soil}	--	--	--	--	0.50	0.44	--	0.32	0.25
IR _{dust, 0–6 years}	0.13	--	0.06	0.19	--	0.02	0.59	--	0.12
IR _{dust, 6–70 years}	0.16	--	0.08	0.24	--	0.04	0.03	--	0.01
IR _{soil, 0–18 years}	--	0.13	0.07	--	0.30	0.26	--	0.66	0.53
IR _{soil, 18–70 years}	--	0.06	0.03	--	0.18	0.15	--	--	--

^a[BaPEQ, benzo[*a*]pyrene equivalents; UA, unsealed asphalt pavement; CSA, coal-tar-sealed pavement; IR, ingestion rate]. ^b-- No contribution to variability is expected from this parameter. ^cBody weight 18–70 years of age contributed ~1% to variability of estimates.

contributed relatively little. When lifetime exposure or exposure only during the first 6 years of life to CSA environments was considered (scenarios 2 and 3), IR contributed a greater proportion of the variability in estimated ELCR.

DISCUSSION

Four exposure scenarios for nondietary ingestion of CSA-affected soil and SHD resulted in estimated BaPEQ doses that are substantially elevated over the dose for urban background (Table 1). BaPEQ doses from nondietary ingestion of CSA-affected soil and dust range from 91 ng/kg/day during the first year of life to 9.1 ng/kg/day for adults. For comparison, Chuang et al. (1999)²⁶ reported dietary intake for the sum of B2 PAHs for children (2–4 years of age) in North Carolina as 24.8 ng/kg/day. Dietary intakes among adults of B2 PAHs have been estimated at between 1 and 5 µg/day on average (about 12.5–62.5 ng/kg/day).⁴⁴ We recently demonstrated that exposures to B2 PAHs in CSA-affected SHD are expected to exceed dietary intakes in children.²⁸

ELCRs associated with CSA-affected settings (scenarios 2–5) greatly exceed those for the urban background (scenario 1). To put CSA-associated ELCRs into context, estimated CTE ELCR for lifetime exposure to CSA-affected soils (8.9×10^{-5}) exceeds that for urban soils in Beijing, China (1.77×10^{-6}),⁴⁵ and CTE ELCR for lifetime exposure to CSA-affected SHD (2.0×10^{-5}) exceeds that for exposure to urban surface dust (pavement and road dust) in an industrial area in China (1.05×10^{-6}).⁴⁶ However, estimated RME ELCR for lifetime exposure to CSA-affected SHD (5.82×10^{-5}) was less than that reported by Maertens et al. (2008)⁴⁷ for children in those residences in Ottawa, Canada, with SHD PAH in the top 10th percentile ($>1 \times 10^{-4}$), although the IR and SHD PAH concentrations were comparable to those used here. The difference likely arises because Maertens et al. included an adjustment factor in their risk analysis to account for exposures taking place during early life stages. ELCRs estimated here for CSA-affected settings exceed those for some other types of exposure to PAHs. For example, estimated CTE ELCRs for CSA-affected settings are much greater than those estimated for ingestion of grilled and smoked meat (2.63×10^{-7})⁴⁸ and for inhalation of granulates associated with intense 30-year activity on artificial turf (1×10^{-6} for presumed worst case conditions).⁴⁹

The increased cancer risk associated with CSA-affecting settings likely affects a large number of people in the U.S. Use of the product is widespread in the U.S. east of the Continental Divide,⁴ and it also is used in some parts of Canada.⁸ Sealed parking lots constituted 1–2% of the area of four mixed

commercial and residential neighborhoods mapped in Texas; in a suburb of Chicago, IL, sealcoated pavement constituted 4% of the area, and 89% of driveway area was sealcoated.¹⁸

Uncertainty. The analysis presented here contains several sources of uncertainty, and many of the choices made for the analysis result in conservative (lower) estimates of ELCR. Concentrations of one of the B2 PAH, diBaA, were not included in computation of BaPEQ because analytical difficulties resulted in nondetections in all but one of the SHD samples.¹⁸ The cancer slope factor used was 7.3; Schneider et al., (2002)⁵⁰ on the basis of oral carcinogenicity studies with BaP and coal-tar mixtures, recommend use of a slope factor of 11.5, which would increase ELCR reported here by about 50%. No adjustment factor was used to account for increased risk associated with exposure during early life stages, when children are more susceptible to the effects of chemical exposures.⁵¹

Although seven carcinogenic PAHs, all of which have a RPF ≤ 1 , were considered here, the USEPA recently has proposed that 24 PAHs, with RPFs ranging from 0.1 to 60, be used to determine the relative potency of PAH mixtures.⁵² At least three of the PAHs with proposed RPFs exceeding 1—benzo[*c*]fluorene, proposed RPF of 30; dibenz[*a,h*]anthracene, proposed RPF of 10; and dibenzo[*a,l*]pyrene, proposed RPF of 30⁵²—are components of coal tar,^{53,54} and BaPEQs associated with coal tar are estimated to increase by almost a factor of 10 if the proposed RPFs are adopted.⁵⁵

Other elements of the analysis also contributed to conservative ELCRs estimates. Most importantly, the risk analysis presented here did not consider nondietary ingestion of outdoor dust on parking lots, driveways, and playgrounds with coal-tar-based sealcoat, as no data are available that quantify IR for these settings. PAH concentrations in dust from coal-tar-sealcoated pavement, however, are 10 or more times higher than those measured in CSA-affected SHD and soil: median BaPEQ concentrations reported range from 60² to 392 µg/g.¹⁸ Ingestion of 4–8 mg of dust from CSA parking lots per day in children less than 6 years of age would add 100 ng BaPEQ/kg/day to the overall dose (data not shown). By comparison, the maximum calculated dose in the CTE scenarios is 91 ng/kg/day.

Further, the BaPEQ concentrations for CSA SHD in the analysis presented here might underrepresent typical BaPEQ associated with CSA-affected environments, because the samples used as representative were collected in Austin in 2008, about 2 1/2 years after use of coal-tar-based pavement sealant was banned in that city.⁵⁶ It is not known if or how rapidly concentrations of PAH in SHD decrease as sealant on

the adjacent pavement ages. Inhalation of gas-phase PAHs also was not considered here, and recent measurements of air concentrations of PAHs indicate relatively high concentrations above old (3.6–8 yr) coal-tar-based sealant³⁸ and very high concentrations above pavement within hours to weeks following sealant application.⁵⁷

Other sources of uncertainty in this risk analysis include choice of IRs, assumption of 100% bioavailability, sample size, and dust loading. Ingestion rate contributed a large proportion of the variability in estimated ELCR associated with CSA-affected settings. For this analysis we used IRs from.^{37,39} Dust IRs recently recommended by the USEPA are higher than those used here, but soil IRs are lower.⁴⁰ Recalculation of risk estimates using those in the 2011 updated version of the Handbook slightly changes risk estimates but does not change the overall conclusions of our assessment. The assumption of 100% bioavailability likely causes moderate overstatement of risks from ingestion of CSA-affected SHD and soil. The bioavailability of PAHs in abraded particles of coal tar-based sealant has not been investigated, and thus the relevance of studies of the bioavailability of BaP and other B2 PAHs in soil may or may not be robustly applicable to these calculations. Our calculations indicate that bioavailability on the order of 20% would still be associated with risk in excess of 1×10^{-4} in some exposure scenarios (RME, scenario 2). Bioavailability of PAHs in soil has been observed to range as high as 90%.²¹

The data set available for PAHs specifically associated with CSA- and UA-affected settings was relatively small. In particular, data from only three soil samples were available for soil adjacent to unsealed asphalt. However, these concentrations are consistent with upper ranges of concentrations reported in the literature as “background.” Sensitivity analysis indicates that the much of the variability in risk estimates arises from concentrations of BaPEQ in SHD and soil (Table 3).

Finally, the data on dust loading adds some uncertainty to the risk estimates. Recall that one data point in the UA SHD data set is an outlier ($883 \mu\text{g}/\text{cm}^2$, compared to a mean of $85 \mu\text{g}/\text{cm}^2$ for the remaining 6 data points). Reanalysis of the set without this data point shows that CSA settings had a significantly higher dust loading than the UA settings ($p = 0.043$, Student's t test). The source of this difference between the sampled settings is unclear.

In this analysis, lifetime estimated ELCRs for deterministic and probabilistic approaches were virtually identical (Tables 1 and 2, Figure 3). This indicates that point estimates for these parameters, as applied here, reasonably represent values in the center and upper reaches of the distributions of these data. Several of the factors contributing to uncertainty associated with the ELCRs presented here could be more fully accounted for with additional data, resulting in less uncertainty. Because the recognition of coal-tar-based pavement sealants as a source of PAHs to the environment is relatively recent (the first study was published in 2004), there are data gaps for such information as bioavailability of PAHs associated with dried sealant particles, IRs for pavement dust, and change in PAH concentrations in CSA-affected soils and SHD with time since sealant application. Additional data on PAH concentrations in CSA-affected soils and SHD will result in more robust ELCR estimates.

Estimates of excess cancer risk arising from exposure to carcinogenic PAHs in settled house dust and soil near coal tar-sealed parking lots exceeded 1×10^{-4} for the central tendency

estimate for lifetime exposure, and for reasonable maximum estimates for all exposure scenarios considered. Exposure to these compounds in settled house dust is a particularly important source of risk for children younger than 6 years of age, as they are expected to ingest this material at higher rates. This indicates that the use of coal-tar-based pavement sealants magnifies aggregate exposures to B2 PAHs in children and adults in residences adjacent to where these products are used, and is associated with human health risks in excess of widely accepted standards. Although the analysis presented here is based on a limited data set, the results indicate that biomonitoring might be warranted to characterize the exposure of children and adults to PAHs associated with coal-tar-based pavement sealant.

■ ASSOCIATED CONTENT

§ Supporting Information

Additional information on dose and exposure assumptions, estimated doses, and dust loading. Table S1. Mass of house dust ($<0.5 \text{ mm}$) collected, area sampled, surface dust loading, and benzo[*a*]pyrene equivalent (BaPEQ) loading for 18 apartments in the Austin, Tex., area. Table S2. Exposure assumptions for deterministic and probabilistic risk calculations. Table S3. Theoretical yearly doses of benzo[*a*]pyrene equivalents under central tendency and reasonable maximum exposure conditions. This material is available free of charge via the Internet at <http://pubs.acs.org>.

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Notes

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■ ABBREVIATIONS:

AT	averaging time
B2 PAH	carcinogenic polycyclic aromatic hydrocarbons (classified B2 by EPA)
BaP	benzo[<i>a</i>]pyrene
CSA	coal-tar-sealed asphalt
CTE	central tendency exposure
BaPEQ	benzo[<i>a</i>]pyrene equivalents
BW	body weight
ED	exposure duration
EF	exposure frequency
ELCR	excess lifetime cancer risk
IR	ingestion rate
PAH	polycyclic aromatic hydrocarbon
RME	reasonable maximum exposure
RPF	relative potency factor
SHD	settled house dust
SHEDS	Stochastic Human Exposure and Dose Simulation
UA	unsealed asphalt

REFERENCES

- (1) Mahler, B. J.; Van Metre, P. C.; Bashara, T. J.; Wilson, J. T.; Johns, D. A. Parking lot sealcoat: An unrecognized source of urban polycyclic aromatic hydrocarbons. *Environ. Sci. Technol.* **2005**, *39* (15), 5560–5566.
- (2) *Polycyclic Aromatic Hydrocarbons Released from Sealcoated Parking Lots—a Controlled Field Experiment to Determine if Sealcoat Is a Significant Source of PAHs in the Environment*; University of New Hampshire Stormwater Center. Final Report; University of New Hampshire Stormwater Center: Durham, NH, 2010.
- (3) Van Metre, P. C.; Mahler, B. J. Contribution of PAHs from coal-tar pavement sealcoat and other sources to 40 U.S. lakes. *Sci. Total Environ.* **2010**, *409* (2), 334–344, DOI: S0048-9697(10)00847-8 [pii] 10.1016/j.scitotenv.2010.08.014.
- (4) Van Metre, P. C.; Mahler, B. J.; Wilson, J. T. PAHs underfoot: contaminated dust from coal-tar sealcoated pavement is widespread in the United States. *Environ. Sci. Technol.* **2009**, *43* (1), 20–25.
- (5) Yang, Y.; Van Metre, P. C.; Mahler, B. J.; Wilson, J. T.; Ligouis, B.; Razaque, M. D.; Schaeffer, D. J.; Werth, C. J. Influence of coal-tar sealcoat and other carbonaceous materials on polycyclic aromatic hydrocarbon loading in an urban watershed. *Environ. Sci. Technol.* **2010**, *44* (4), 1217–1223, DOI: 10.1021/es902657h.
- (6) Mahler, B. J.; Van Metre, P. C.; Wilson, J. T. Concentrations of polycyclic aromatic hydrocarbons (PAHs) and major and trace elements in simulated rainfall runoff from parking lots, Austin, Texas, 2003 (version 3); U.S. Geological Survey Open-File Report: 2004–1208, p. 87.
- (7) Scoggins, M.; Ennis, T.; Parker, N.; Herrington, C. A photographic method for estimating wear of coal tar sealcoat from parking lots. *Environ. Sci. Technol.* **2009**, *43* (13), 4909–4914.
- (8) Diamond Environmental Group. Reconnaissance study of coal tar sealcoat application in Toronto and an estimate of related PAH emissions. Departments of Geography and Chemical Engineering, University of Toronto, 2011.
- (9) National Institutes of Health. Coal tars and coal-tar pitches. National Institute of Environmental Health Science, National Institutes of Health, Department of Health and Human Services, 2011.
- (10) Kaushik, S.; Rainal, R. K.; Bhatia, G.; Verma, G.; Khandal, R. K. Modification of coal tar pitch by chemical method to reduce benzo(a)pyrene. *Curr. Sci.* **2007**, *93* (4), 540–544.
- (11) *Water: CWA Methods, Priority Pollutants*; <http://water.epa.gov/scitech/methods/cwa/pollutants.cfm>.
- (12) Mahler, B. J.; Metre, P. C.; Crane, J. L.; Watts, A. W.; Scoggins, M.; Williams, E. S. Coal-tar-based pavement sealcoat and PAHs: implications for the environment, human health, and stormwater management. *Environ. Sci. Technol.* **2012**, *46* (6), 3039–3045.
- (13) Van Metre, P. C.; Mahler, B. J.; Furlong, E. T. Urban sprawl leaves its PAH signature. *Environ. Sci. Technol.* **2000**, *34* (19), 4064–4070, DOI: 10.1021/es991007n.
- (14) Bryer, P. J.; Scoggins, M.; McClintock, N. L. Coal-tar based pavement sealant toxicity to freshwater macroinvertebrates. *Environ. Pollut.* **2010**, *158* (5), 1932–1937, DOI: 10.1016/j.envpol.2009.10.038.
- (15) Scoggins, M.; McClintock, N. L.; Gosselink, L.; Bryer, P. Occurrence of polycyclic aromatic hydrocarbons below coal-tar-sealed parking lots and effects on stream benthic macroinvertebrate communities. *J. North Am. Benthol. Soc.* **2007**, *26* (4), 694–707.
- (16) Bommarito, T.; Sparling, D. W.; Halbrook, R. S. Toxicity of coal-tar pavement sealants and ultraviolet radiation to *Ambystoma maculatum*. *Ecotoxicology* **2010**, *19* (6), 1147–1156, DOI: 10.1007/s10646-010-0498-8.
- (17) Bryer, P. J.; Elliott, J. N.; Willingham, E. J. The effects of coal tar based pavement sealer on amphibian development and metamorphosis. *Ecotoxicology* **2006**, *15* (3), 241–247, DOI: 10.1007/s10646-005-0055-z.
- (18) Mahler, B. J.; Metre, P. C.; Wilson, J. T.; Musgrove, M.; Burbank, T. L.; Ennis, T. E.; Bashara, T. J. Coal-tar-based parking lot sealcoat: An unrecognized source of PAH to settled house dust. *Environ. Sci. Technol.* **2010**, *44* (3), 894–900, DOI: 10.1021/es902533r.
- (19) Agency for Toxic Substances and Disease Registry. Toxicological profile for polycyclic aromatic hydrocarbons. Atlanta, GA, U.S. Department of Health and Human Services, Public Health Service, 1995.
- (20) Hoffmann, D.; Hoffmann, I. The changing cigarette, 1950–1995. *J. Toxicol. Environ. Health.* **1997**, *50* (4), 307–364, DOI: 10.1080/009841097160393.
- (21) Ramesh, A.; Walker, S. A.; Hood, D. B.; Guillen, M. D.; Schneider, K.; Weyand, E. H. Bioavailability and risk assessment of orally ingested polycyclic aromatic hydrocarbons. *Int. J. Toxicol.* **2004**, *23* (5), 301–333, DOI: WGMXSTX4L3U8CJF7 [pii] 10.1080/10915810490517063.
- (22) Jones-Otazo, H. A.; Clarke, J. P.; Diamond, M. L.; Archbold, J. A.; Ferguson, G.; Harner, T.; Richardson, G. M.; Ryan, J. J.; Wilford, B. Is house dust the missing exposure pathway for PBDEs? An analysis of the urban fate and human exposure to PBDEs. *Environ. Sci. Technol.* **2005**, *39* (14), 5121–5130.
- (23) Lioy, P. J.; Freeman, N. C.; Millette, J. R. Dust: A metric for use in residential and building exposure assessment and source characterization. *Environ. Health Perspect.* **2002**, *110* (10), 969–983, DOI: sc271_5_1835 [pii].
- (24) Maertens, R. M.; Bailey, J.; White, P. A. The mutagenic hazards of settled house dust: A review. *Mutat. Res.* **2004**, *567* (2–3), 401–425, DOI: S1383-5742(04)00061-4 [pii] 10.1016/j.mrrev.2004.08.004.
- (25) Whitehead, T.; Metayer, C.; Gunier, R. B.; Ward, M. H.; Nishioka, M. G.; Buffler, P.; Rappaport, S. M. Determinants of polycyclic aromatic hydrocarbon levels in house dust. *J. Expo. Sci. Environ. Epidemiol.* **2011**, *21* (2), 123–132, DOI: jes200968 [pii] 10.1038/jes.2009.68.
- (26) Chuang, J. C.; Callahan, P. J.; Lyu, C. W.; Wilson, N. K. Polycyclic aromatic hydrocarbon exposures of children in low-income families. *J. Expo. Anal. Environ. Epidemiol.* **1999**, *9* (2), 85–98.
- (27) Wilson, N. K.; Chuang, J. C.; Lyu, C.; Menton, R.; Morgan, M. K. Aggregate exposures of nine preschool children to persistent organic pollutants at day care and at home. *J. Expo. Anal. Environ. Epidemiol.* **2003**, *13* (3), 187–202, DOI: 10.1038/sj.jea.7500270 [pii].
- (28) Williams, E. S.; Mahler, B. J.; Van Metre, P. C. Coal-tar pavement sealants might substantially increase children's PAH exposures. *Environ. Pollut.* **2012**, *164*, 40–41 DOI:.
- (29) U.S. Environmental Protection Agency. Risk Assessment Guidance for Superfund Volume I, Human Health Evaluation Manual (Part A). EPA/540/1-89/002; Office of Research and Development: Washington, DC, 1989.
- (30) U.S. Environmental Protection Agency. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. EPA/600/R-93/089; Office of Research and Development: Washington, DC, 1993.
- (31) Nam, J. J.; Sweetman, A. J.; Jones, K. C. Polynuclear aromatic hydrocarbons (PAHs) in global background soils. *J. Environ. Monit.* **2009**, *11* (1), 45–48, DOI: 10.1039/b813841a.
- (32) Lemieux, C. L.; Lambert, I. B.; Lundstedt, S.; Tysklind, M.; White, P. A. Mutagenic hazards of complex polycyclic aromatic hydrocarbon mixtures in contaminated soil. *Environ. Toxicol. Chem.* **2008**, *27* (4), 978–990, DOI: 07-157 [pii] 10.1897/07-157.1.
- (33) Turczynowicz, L.; Fitzgerald, D. J.; Nitschke, M.; Mangas, S.; McLean, A. Site contamination health risk assessment case study involving tenant relocation from a former gasworks site. *J. Toxicol. Environ. Health A* **2007**, *70* (19), 1638–1653, DOI: 781628196 [pii] 10.1080/15287390701434737.
- (34) Ozkaynak, H.; Xue, J.; Zartarian, V. G.; Glen, G.; Smith, L. Modeled estimates of soil and dust ingestion rates for children. *Risk Anal.* **2010**, *31* (4), 592–608, DOI: 10.1111/j.1539-6924.2010.01524.x.
- (35) *Consolidated Human Activity Database*; (<http://www.epa.gov/chadnet1/>).
- (36) Harrad, S.; Ibarra, C.; Diamond, M.; Melymuk, L.; Robson, M.; Douwes, J.; Roosens, L.; Dirtu, A. C.; Covaci, A. Polybrominated

- diphenyl ethers in domestic indoor dust from Canada, New Zealand, United Kingdom and United States. *Environ. Int.* **2008**, *34* (2), 232–238, DOI: S0160-4120(07)00160-2 [pii]10.1016/j.envint.2007.08.008.
- (37) *Exposure Factors Handbook (Final Report)*; U.S. Environmental Protection Agency: Washington, D.C., 1997.
- (38) U.S. Environmental Protection Agency. *Child-Specific Exposure Factors Handbook*. Washington, DC, USEPA, 2008.
- (39) Van Holderbeke, M.; Cornelis, C.; Bierkens, J.; Torfs, R. Review of the soil ingestion pathway in human exposure assessment. VITO/RIVM. Flanders, Belgium, VITO/RIVM, 2008.
- (40) U.S. Environmental Protection Agency. *Exposure Factors Handbook*, 2011 ed.; Washington, DC, USEPA, 2011.
- (41) Portier, K.; Tolson, J. K.; Roberts, S. M. Body weight distributions for risk assessment. *Risk Anal.* **2007**, *27* (1), 11–26, DOI: 10.1111/j.1539-6924.2006.00856.x.
- (42) HH: *Risk Characterization, Region 8*; <http://ehp03.niehs.nih.gov/static/instructions.action#type>.
- (43) *Integrated Risk Information System, Benzo[a]pyrene (BaP)* (CASRN 50–32–8); <http://www.epa.gov/iris/subst/0136.htm>.
- (44) Menzie, C. A.; Potocki, B. B.; Santodonato, J. Exposure to Carcinogenic PAHs in the Environment. *Environ. Sci. Technol.* **1992**, *26* (7), 1278–1284.
- (45) Peng, C.; Chen, W.; Liao, X.; Wang, M.; Ouyang, Z.; Jiao, W.; Bai, Y. Polycyclic aromatic hydrocarbons in urban soils of Beijing: Status, sources, distribution and potential risk. *Environ. Pollut.* **2011**, *159* (3), 802–808, DOI: S0269-7491(10)00511-7 [pii]10.1016/j.envpol.2010.11.003.
- (46) Wang, W.; Huang, M. J.; Kang, Y.; Wang, H. S.; Leung, A. O.; Cheung, K. C.; Wong, M. H. Polycyclic aromatic hydrocarbons (PAHs) in urban surface dust of Guangzhou, China: Status, sources and human health risk assessment. *Sci. Total Environ.* **2011**, *409* (21), 4519–4527, DOI: S0048-9697(11)00748-0 [pii]10.1016/j.scitotenv.2011.07.030.
- (47) Maertens, R. M.; Yang, X.; Zhu, J.; Gagne, R. W.; Douglas, G. R.; White, P. A. Mutagenic and carcinogenic hazards of settled house dust. I: Polycyclic aromatic hydrocarbon content and excess lifetime cancer risk from preschool exposure. *Environ. Sci. Technol.* **2008**, *42* (5), 1747–1753.
- (48) Alomirah, H.; Al-Zenki, S.; Husain, A.; Sawaya, W.; Ahmed, N.; Gevao, B.; Kannan, K. Benzo[a]pyrene and total polycyclic aromatic hydrocarbons (PAH) levels in vegetable oils and fats do not reflect the occurrence of the eight genotoxic PAHs. *Food Addit. Contam. Part A: Chem. Anal. Control Expo. Risk Assess* **2010**, *27* (6), 869–878.
- (49) Menichini, E.; Abate, V.; Attias, L.; De Luca, S.; di Domenico, A.; Fochi, I.; Forte, G.; Iacovella, N.; Iamiceli, A. L.; Izzo, P.; Merli, F.; Bocca, B. Artificial-turf playing fields: Contents of metals, PAHs, PCBs, PCDDs and PCDFs, inhalation exposure to PAHs and related preliminary risk assessment. *Sci. Total Environ.* **2011**, *409* (23), 4950–4957, DOI: S0048-9697(11)00760-1 [pii]10.1016/j.scitotenv.2011.07.042.
- (50) Schneider, K.; Roller, M.; Kalberlah, F.; Schuhmacher-Wolz, U. Cancer risk assessment for oral exposures to PAH mixtures. *J. Appl. Toxicol.* **2002**, *22* (1), 73–83.
- (51) World Health Organization. Principles for evaluating health risks in children associated with exposure to chemicals. Geneva, Switzerland, 2006.
- (52) U.S. Environmental Protection Agency. Development of a relative potency factor (RPF) approach for polycyclic aromatic hydrocarbon (PAH) mixtures. Washington, DC, 2010.
- (53) Agency for Toxic Substances and Disease Registry. Toxicological profile for creosote. Atlanta, GA, U.S. Department of Health and Human Services, Public Health Service, 2002.
- (54) Wise, S. A.; Poster, D. L.; Leigh, S. D.; Rimmer, C. A.; Mossner, S.; Schubert, P.; Sander, L. C.; Schantz, M. M. Polycyclic aromatic hydrocarbons (PAHs) in a coal tar standard reference material–SRM 1597a updated. *Anal. Bioanal. Chem.* **2010**, *398* (2), 717–728, DOI: 10.1007/s00216-010-4008-x.
- (55) Rohr, A. C. Comments on development of a relative potency factor (RPF) approach for polycyclic aromatic hydrocarbon (PAH) mixtures, external review draft. Electric Power Research Institute: Palo Alto, CA, 2010.
- (56) City of Austin. An ordinance amending the city code to add a new chapter 6–6 relating to coal tar pavement products, creating offenses, and providing penalties. 2051117–070. Austin, Texas, 2005.
- (57) Van Metre, P. C.; Majewski, M. S.; Mahler, B. J.; Foreman, W. T.; Braun, C. L.; Wilson, J. T.; Burbank, T. L. PAH volatilization following application of coal-tar-sealed pavement. *Atmos. Environ.* **2012**, *51*, 108–115.
- (58) Van Metre, P. C.; Majewski, M. S.; Mahler, B. J.; Foreman, W. T.; Braun, C. L.; Wilson, J. T.; Burbank, T. L. Volatilization of polycyclic aromatic hydrocarbons from coal-tar-sealed pavement. *Chemosphere* **2012**, *88* (1), 1–7, DOI: S0045-6535(11)01466-4 [pii]10.1016/j.chemosphere.2011.12.072.